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EXPERIMENTAL ACCLIMATIZATION TO THE TROPICAL SUN [1]

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INTRODUCTION

After a series of exposures of monkeys to the sun, Aron [2] concluded that the monkey was more susceptible to the action of the sun than any other animal with which he was acquainted, "including even the white man," and that monkeys exposed to the sun in a garden or on a roof in Manila die within seventy or eighty minutes, even though exposed in the early forenoon, in the coolest months of the year.

If this be true, the monkey ought to be an especially favorable subject for a study of acclimatization. I began such a study in October, 1911.

During the course of the experiments four objects were kept in view: (1) To see if the monkey gradually exposed to the sun would undergo a change which would enable it to live in the sun throughout a hot day; (2) if this change should take place, to learn its nature; (3) to determine the relative importance of the various meteorological factors which may combine to influence acclimatization; (4) to learn in how far extraneous factors, such as work, excitement, clothing, drugs, diet, and disease, may influence the course of events.

The monkeys used in this study were of the kind commonly obtainable in Manila. They are of two sizes and may be of two species. Probably both belong to the genus *Pithecus*.[3]

[1] Received for publication September 29, 1916. Read before the Philippine Islands Medical Association, November, 1912.

[2] *This Journal, Sec. B* (1911), 6, 110, 130.

[3] For the scientific names of Philippine monkeys, see Hollister, *This Journal, Sec. D* (1912), 7, 37; and *Proc. U. S. Nat. Mus.* (1914), 46, 328.

Experiments have been made almost daily for more than six months, including the hottest month known in Manila in six years (April, 1912). Twenty-three monkeys have been employed thus far.

The method of study was as follows: After a preliminary study of the variation of the monkey's body temperature in the shade, the monkey was placed in the sun rather early in the morning, and during the exposure, frequent observations were made of the pulse, respiration, and body temperature. Other symptoms were also looked for, such as position assumed, moisture of the skin, and signs of discomfort. Observations were also made upon the temperature of the atmosphere surrounding the monkey and the temperature of the surface upon which or near which it rested. The humidity of the atmosphere, reading of the black-bulb thermometer, and the amount of sunshine, as well as the velocity of the wind, were also noted. Monkeys were placed on the ground, on the asphalt roof of the laboratory, or upon slender poles raised above these surfaces. During the experiments the monkeys were kept upon a constant diet that seemed to be suitable, and they were offered water at frequent intervals. They were weighed daily. They were handled with great gentleness, and even the wildest soon ceased to show signs of fear. Overnight they were kept in clean cages.

EXPERIMENTAL

MONKEY 4

Monkey 4 seemed to show in the sun the typical reaction of a young, healthy Philippine monkey. Immediately below is a brief account of the main happenings in its experience during five months in the Manila sun.

This was a strong, active, healthy, wild male, which we surmised to be rather young. It weighed 1,528 grams when obtained, but within a few days it lost 100 grams. It has maintained practically constant weight ever since. We have not been able to detect in this monkey any signs of sickness. It has never suffered any significant injury. During the course of the experiments it has become much tamer.

A simple, wholesome diet, consisting of boiled white rice and raw ripe banana, was chosen. Twenty-five grams of each food for each kilogram of body weight were furnished the monkey at about the same hour once each day. Slight temporary changes in the diet from time to time did not seem to change the results.

At frequent intervals the monkey was given all the water it would drink.

The body temperature was taken at intervals through the day with a clinical thermometer, pushed well into the rectum and kept in place until there was no further rise. The monkey was excited as little as possible.

Temperature of the monkey in the shade.—The observations on this monkey's temperature were begun on the afternoon of November 27. The monkey was at first kept in the shade in the animal house (iron roof 3 to 5 meters high, no ceiling, well ventilated through netting). On November 28 the temperature both of the monkey and of the surrounding air was observed. The results of the observations are given in Table I.

TABLE I.—*Temperature of monkey 4 in shade in the animal house, November 28, 1911.*

Time.	Air temperature in house.	Body temperature.
a. m.	°*C.*	°*C.*
8.20	25	38.0
10.00	30	38.0
11.00	31	38.6
p. m.		
1.42	31.7	38.9
2.40	32	39.0
3.40	30	39.3
4.20	30.5	39.2

From Table I it is seen: (1) That there may be a rather marked rise in the body temperature of monkeys kept in the shade; that the body temperature began to rise between 10 and 11 o'clock, after the air temperature had reached 30° C., that is, after the air temperature had already risen 5°; (2) that, although the monkey remained in the sun throughout the day, about one half of the rise in body temperature occurred between 10 and 11 o'clock; (3) that it continued to rise gradually until 3.40 in the afternoon; (4) that between 2.40 and 3.40 in the afternoon the rise in body temperature was accompanied by a fall in air temperature; (5) that the maximum body temperature (39.3°) was apparently one hour later than the maximum air temperature (32°); (6) that the body temperature fell more slowly than the air temperature; (7) that this monkey's maximal normal body temperature is probably not below 39.5°, an observation in accord with the findings of Simpson and

Galbraith [4] for two other species of monkeys; (8) that there is apparently a wide variation in this monkey's normal temperature. This variation in body temperature seems to indicate, as has been observed in other monkeys, that the temperature-regulating mechanism of the monkey is not as efficient as is that of man. This suggests the possibility that, if the monkey can become adapted to life in the "tropical sun," man could more readily become adapted. The responsiveness of the monkey's temperature to external influences also suggests that the change in body temperature of an unacclimatized monkey might be a good index of the action of the sun upon the monkey and, possibly, an index of the sun's harmfulness for other animals, including man.

The monkey is not injured by being seven hours in the sun.—The first experimental exposure of this monkey to the sun was made on November 29, 1911. It was exposed to the sun at 8.35 in the forenoon on a horizontal pole resting 1 meter above a "skin" tennis court. It was free to walk back and forth. It received food at the usual hour, and water was given after taking the temperature.

The amount of sunshine recorded on this day at the Weather Bureau building, 0.3 kilometer distant, was nine hours and six minutes. If we take the Weather Bureau records, which would be approximately accurate for the tennis court, as a basis for our estimate, this monkey was exposed to the sun for five hours and twelve minutes before any clouds appeared, that is, from 8.35 in the forenoon to 1.47 in the afternoon. The clouds lasted eight minutes; the monkey was again subjected to the action of the sun, this time for one hour and twenty-four minutes. At 3.20 in the afternoon it had a respite from the sun of seven minutes and was afterward in the sun for forty-eight minutes, until 4.15 in the afternoon. This makes it probable that on this day the monkey was exposed to the Manila sun for about seven and one-half hours, including the hottest portion of the day.

The monkey showed no symptoms of distress from the action of the sun, although exposed for seven hours. It did, however, show some depression, as it lay on the pole for short periods in the more severe portions of the day. No sign of injury was noticed on this day or on any succeeding day.

The effect of the sun on the monkey's temperature.—Although the monkey showed no distress from this long exposure to the

[4] *Trans. Roy. Soc. Edin.* (1906), 45, I, 65–104. The maximum normal temperature of *Rhesus macacus* observed by Eyre and Kennedy was 40°. *Journ. Physiol.* (1907), 35, xxx-xxxi.

sun, yet there was a distinct measureable effect produced. Table II shows the effect of the sun upon this monkey as indicated by body temperature.

TABLE II.—*Effect of sun upon monkey's temperature. Monkey 4 in the sun, 1 meter above the ground, November 29, 1911.*

Time.	Air temperature.[a]	Body temperature.	Time.[b]	Black-bulb reading.	Wind. Meters per second.[c]	Relative humidity.	Remarks.
a. m.	°C.	°C.	*a. m.*	°C.		*P. ct.*	
8.10	25	38					In animal house.
8.35	28						Exposed to sun at 8.35 a. m.
9.28	29	39.7	9.00	[d]48.2	0.0	59.0	After 53 minutes' exposure. No distress symptoms.
10.25	30	40.8	10.00	[d]51.7	2.0	56.0	
11.25	30.2	39.5	11.00	47.7	3.0	56.0	No distress symptoms.
			12.00	[d]47.3	3.5	55.0	Do.
p. m.			*p. m.*				
1.15	32	39.4	1.00	[d]50.9	4.0	51.5	Do.
2.15	32	40.0	2.00	50.0	0.0	48.0	8 minutes of cloud (1.47–1.55), no distress symptoms.
3.20	31	39.6	3.00	[d]47.2	3.0	46.0	No distress symptoms.
4.15	30	39.4	4.00	[d]44.4	0.0	50.0	7 minutes of cloud (3.20–3.27), no distress symptoms.

[a] The air temperature here given was that registered by an ordinary chemical thermometer hanging in the sun near the monkey.

[b] Time of Weather Bureau observation.

[c] Wind velocity in meters per second.

[d] Interpolations. Since the black-bulb readings at the Weather Bureau are taken at relatively infrequent intervals, a method of obtaining interpolations recommended by the Weather Bureau authorities was adopted. It is as follows:

"Find the difference between black-bulb readings and the corresponding readings of the Violle actinometer. Interpolate as many means between these differences as there are observation times between the black-bulb readings. Substract or add these means as the case demands to the corresponding Violle readings to get the black-bulb interpolations. '

The method is illustrated by the following table:

Time.	Black-bulb.	Violle.	Difference.	Mean.
a. m.	°C.	°C.	°C.	°C.
8.00	42.5	53.0	−10.5	
9.00	[a](48.2)	59.0	----------	−10.76
10.00	(51.7)	62.7	----------	−11.02
11.00	47.7	59.0	−11.3	
12.00	(47.3)	58.5	----------	−11.2
p. m.				
1.00	(50.9)	62.0	----------	−11.1
2.00	(50.0)	61.0	−11.0	
3.00	(47.2)	60.0	----------	−12.8
4.00	(44.4)	59.0	----------	14.6
5.00	27.6	44.0	−16.4	

[a] The interpolations are inclosed in parentheses.

Table II shows that the temperature of the monkey did not rise above 40.8° on seven hours' exposure to the sun. This was rather surprising in view of Aron's statement, and is all the more noteworthy in view of the fact that on this day the monkey, being still wild, struggled hard every time the temperature was taken. On comparing the conditions at 10.25 in the forenoon on November 29 with the conditions at 3.40 in the afternoon on November 28, it is seen that the air temperatures were the same on the two occasions, while the body temperature was 1.5° higher in the sun than in the shade. This difference might be taken for a measure of the effect of the sun's rays, if we did not know that the monkey's temperature depends also upon the air humidity, wind velocity, and individual resistance, factors apparently overlooked by Aron in the statements cited.

Weather data for these two days are given in Table III in a form convenient for comparison. A study of the table will give an idea of the action of the sun's rays alone.

TABLE III.—*Action of the sun's rays alone on monkey 4.*

Exposure.	Date.	Time.	Body temperature.	Time.	Weather.			
					Air temperature.	Black-bulb reading.	Wind. Meters per second.	Humidity.
			°C.	*a. m.*	°C.	°C.		
Sun	Nov. 29	10.25	40.8	10.00	28.8	51.7	2.0	56
				p. m.				
Shade	Nov. 28	3.40	39.3	3.00	30.0	shade	1.0	55
		------	-------	4.00	29.5	shade	0.0	59

When we compare the air temperatures, winds, and humidities of these two periods of observation, we find that, when the monkey was in the sun, the air temperature and humidity were less and the wind was greater than when it was in the shade. Each of these factors—the lower air temperature, the lower humidity, and the stronger wind—would tend to produce a lower temperature in the monkey. Since this was the first exposure to heat in the sun, the power of resistance of the monkey to the sun and heat was probably about the same as on the day previous in the shade. The sun's rays, therefore, are the only factor that would tend to raise the temperature of the monkey above that of the previous day when the monkey was in the shade. There-

fore it seems reasonable to suppose that the increase of 1.5° in body temperature over that of the day before in the shade was due to the sun's rays alone. Since the black-bulb thermometer reading, when the monkey's temperature was raised by the sun's rays, was 51.7°, and since the air temperature was lower at this time, the humidity probably lower, and the wind higher, it would appear that the sun's rays which were strong enough to produce a black-bulb reading of 51.7° were more than strong enough at this time to raise this monkey's body temperature through 1.5°C.

The effect of conditions other than the sun's rays and air temperature upon this monkey.—Table II further shows that the body temperature of this monkey in the sun was higher when the air temperature was only 30° (10.25 in the morning) than when the air temperature was 32° (1.15 or 2.15 in the afternoon). This emphasizes the well-known fact that the hottest weather is not necessarily the most injurious, unless we understand by "hottest" something else than the highest temperature registered by the ordinary thermometer, that is, the atmospheric temperature. If we compare the weather conditions at 10.00 o'clock in the morning, which were soon followed by the highest body temperature observed on this day, with those obtaining at 1.00 in the afternoon, which were closely followed by a body temperature 1.4° lower, we find that this lower body temperature, in spite of a higher air temperature, was accompanied by a lower reading of the black-bulb thermometer, a higher wind velocity, and a lower relative humidity. Each of the last three conditions would work toward offsetting the effect of increased air temperature, and this fact accounts, at least partially, for the lower body temperature when the surrounding air was 2° hotter. These observations show why the portion of the day that was the hottest, judged by the ordinary thermometer, was not the most injurious to the monkey; it was because the energy of the sun's rays was less, the wind was blowing harder, and the air was not so humid. Therefore there are four factors that must always be taken into account in any attempt to determine the cause of a rise in body temperature or of death following exposure to the sun, namely, the energy of the sun's rays and the temperature, the movement, and the humidity of the air.

The relation of heat to the death of monkeys in the sun.—Since Aron makes the statement that monkeys die in "seventy to eighty

minutes" when exposed to the Manila sun, even in the coolest portion of the year,[5] and since this monkey did not die, though exposed to the Manila sun for seven hours and twenty-four minutes, it might be inferred that this day happened to be exceptionally cool, much cooler than the days on which Aron's monkeys died. On comparing the Weather Bureau records for these days, however, the energy of the sun rays, to which this monkey was exposed, was greater and the surrounding atmosphere was hotter than those to which Aron's monkeys were exposed. The air temperature and the black-bulb readings are given in Table IV.

TABLE IV.—*Relation of sun's rays and atmospheric temperature to the death of monkeys in the sun.*

Monkey No.	Date.	Maximum black-bulb.[a]	Air temperature.	Authority.	Duration of exposures.	Result.
	1910.	°C.	°C.		*H. m.*	
2	Nov. 16	51.8	30.8	Aron[b]	0 38	Death.
	1911.					
11	Jan. 18	43.3	29.8	----do[b]	1 50	Do.
[c]12	Jan. 18	47.3	30.3	----do[b]	0 40	Do.
13	Jan. 26	42.4	28.3	----do[d]	1 0	Do.
4	Nov. 29	51.7	28.8-31.2	Shaklee	7 24	No distress.

[a] Interpolations. For method, see footnote to Table II.
[b] Aron, op. cit., p. 111.
[c] Number 12 was shaved.
[d] Aron, op. cit., p. 114.

From Table IV one might draw the conclusion that the energy of the sun's rays is a relatively small factor among those that combine to produce the monkey's death. It is seen from the table that one monkey remained in the sun seven hours and twenty-four minutes without any signs of distress and that four other monkeys in the sun at the same temperature, or a lower one, and in sun rays of equal or less energy, died in thirty-eight, one hundred ten, forty, and sixty minutes, respectively. This further emphasizes the importance of the conditions other than the temperature of the atmosphere or energy of the sun's rays which act upon the monkeys exposed in the way in which Aron exposed his monkeys. The data given in Table V shows this more clearly.

[5] Aron, *This Journal, Sec. B* (1911), 6, 110.

TABLE V.—*Relation of heat from the ground,*[a] *of the wind, and of humidity to the monkey's death in the sun.*

Monkey of—	Date.	Time of death.	Time of weather observation.	Black-bulb.	Wind.[c]	Relative humidity.	Duration of exposure.	Remarks.
Aron:	1910.	*a. m.*	*a. m.*	°*C.*	*M. s.*	*P. ct.*	*H. m.*	
2	Oct. 16	10.08	10.00	51.8	0 0	69	0 38	On hot surface.
	1911.	*p. m.*	*p. m.*					
11	Jan. 18	4.15	4.00	43.3	3 0	63	1 50	Do.
[b]12	Jan. 18	3.10	3.00	47.3	3 5	61	0 45	Do.
		a. m.	*a. m.*					
13	Jan. 26	11.50	12.00	42.4	3 5	66	1 0	Do.
Shaklee:								
4	Nov. 29	----------	10.00	51.7	2 0	56.0	1 25	Not on hot surface.
			p. m.					
4 (continued).	----------	----------	1.00	50.9	4 0	51.5	4 25	Do.
4(concluded.	----------	Lived	4.00	44.4	0 0	50.0	7 15	Do.

[a] Heat from ground is not shown in this table.

[b] No. 12 was shaved.

[c] Velocity in meters per second.

NOTE: Reports for monkeys 2, 11, and 12 were taken from page 111 of Aron's paper, and for monkey 13 from page 114 of the same paper.

It will be seen from Table V that the heat from the sun's rays as indicated by the black-bulb thermometer was considerably greater in the case of the monkey that lived than in the cases of three that died. Therefore it cannot be said that the sun's rays killed Aron's monkeys. The wind conditions were as favorable to the monkeys that died, but the humidity of the air surrounding the monkeys that died was much greater. There is another important factor in the death of Aron's monkeys which must not be overlooked. His monkeys rested on the ground or roof. The ground and roofs become very hot in a hot sun. It seems possible that the heat which the monkey's body received from the hot roof or from the ground may have far exceeded that absorbed from the sun's rays.

Since the sun's rays combined with the other weather conditions produced so little effect upon monkey 4, in absence of the heat from the ground, it was decided to place the monkey on the ground, to determine the influence of the heat from the ground.

The effect of the combined action of the sun and the heat radiated and conducted from the ground.—On Friday, December

1, the monkey was placed on the tennis court chained to a small scantling, 6.5 by 7.5 centimeters, that was lying on the ground. He was free to move about and to sit on the scantling, which would not be so hot and which would raise him about 6.5 centimeters above the ground. He was put out at 8.25 in the forenoon. This day was also a bright day. The Weather Bureau records show seven hours and eighteen minutes of sunshine. Taking these records again as a basis for our estimate, he was exposed to the sun and hot ground from 8.25 until 11.01 (two hours and twenty-four minutes), from 11.25 to 11.45, and from 12.05 to 2.45 (two hours and twenty-eight minutes). Table VI shows the effects of these conditions upon the monkey's temperature.

TABLE VI.—*Combined action of sun and hot ground on the monkey. Monkey 4, in the sun and on the ground, December 1, 1911.*

Time.	Air temperature.[a]			Body temperature.	Remarks.
	On ground.	8.5 cm. above ground.	1 m. above ground.		
	°C.	°C.	°C.	°C.	
7.55	--------	--------	22.5	37.4	Shade (in the house).
8.25	--------	--------	--------	--------	Exposure began, sun.
9.43	38	32	28.5	39.8	Sunshine.[b]
10.55	42	32.5	29	39.4	Do.[b]
1.17	46	35	32	40.5	Do.[b]
2.30	44.5	35	32.5	41.7	Do.[b]
3.15	37.5	30.5	29	42.0	Cloud.[b]
4.00	34	29.5	29	39.0	Do.[b]

[a] Air temperature as indicated by chemical thermometers kept in the sun in the positions indicated—namely, on the ground, 8.5 centimeters above the ground, and 1 meter above the ground.

[b] Sunshine and cloud as recorded at the Weather Bureau, 0.3 kilometer away.

It will be seen from Table VI that the combined action of the sun and the hot ground for two hours and twenty minutes in the forenoon (8.25–10.55) did not raise the temperature of this monkey above its maximum normal. If monkeys "die in seventy to eighty minutes" when placed on the ground in the Manila sun, this monkey had already acquired a marked increase in powers of resistance; but since not all days are alike, even in Manila, it is necessary to study the meteorological conditions before drawing a conclusion. On December 1, the day of the exposure under consideration, the conditions seemed to be as severe as those obtaining when some of Aron's monkeys died. This is brought out by a comparison of Table VII with Table V.

TABLE VII.—*Weather conditions on December 1, 1911 (at Weather Bureau, 0.3 kilometer away).*

Time.	Black-bulb reading.	Wind.	Relative humidity.	Sunshine (heliograph reading).
a. m.	°C.	*M. s.*	°C.	
8.00	40.5			
9.00	[a]45.8	0 0	62.0	7.31–11.01
10.00	[a]47.8	2 5	60.0	
11.00	48.3	5 0	59.0	
12.00	[a]46.7	3 0	54.0	11.25–11.45 11.46–11.47
p. m.				
1.00	[a]51.7	0 5	58.0	12.05– 2.45
2.00	50.0	1 5	58.0	
3.00	[a]49.8	0 0	50.0	
4.00	[a]36.1	2 0	68.0	3.58– 4.10

[a] Interpolations, see Table II, footnote.

From a comparison of these two tables it seems probable that at 9 o'clock and at 10 o'clock in the forenoon and at 1 o'clock in the afternoon on December 1 the conditions to which monkey 4 was subjected were as severe as those that produced the deaths of monkeys 11 and 13 of Aron's series. If this be the case, the few exposures of monkey 4 had already developed in it a greatly increased capacity for resisting the evil influences of the exposure to the tropical sun under the conditions named. And although it is probable that the wounds in Aron's monkeys reduced their resistance and that Aron's monkeys struggled more because of the insertion of the thermocouple into these inflamed wounds, it seems justifiable to draw the conclusion that No. 4 had already acquired considerable increase in its powers of resistance, especially when it is remembered that it showed no distress at all on this day, nor did it show on the following days any evidences of having received any injury from this exposure. Another possibility is that this monkey was an especially resistant monkey. This does not seem probable, because similar results were obtained with all the monkeys studied in this series as is brought out below.

The progress and degree of acclimatization.—The results of this day's exposure gave me more hope of being able to learn, by the use of the monkey, something about acclimatization, if not to the sun's rays, at least to the combination of all the climatic factors, including the heat from the ground.

Exposures of this monkey were made on every sunny day thereafter, except holidays, for five months. All the later ex-

posures were made on the black asphalt roof of the laboratory. On the first days, during the exposures, the monkey was chained to a scantling which rested upon the roof, but in the last and hottest months it was chained to a wire, so that it rested directly upon the roof. Table VIII, which gives the highest daily maximum temperatures observed in this monkey in the five months, brings out the progress and degree of acclimatization of this monkey to the combined meteorological factors plus the intense heat from the roof—in other words, to the Manila climate at its worst.

TABLE VIII.—*Highest daily maximal body temperatures. Acclimatization (monkey 4 kept in the sun and in contact with the heated roof).*

1911.	°C.
December 1	42.0
December 6, 15	40.5
December 7	40.0
December 12	40.0
December 18	40.0
Average [a]	40.6
December 20	38.5
December 21	39.8
December 22	39.4
December 27	39.3
December 29	39.9
Average	39.6
1912.	
January 3	39.7
January 5, 11, 16	39.5
January 17	39.9
January 26	39.9
January 29	39.9
Average	39.8
February 9	39.5
February 10, 13	39.4
February 12, 27	39.3
February 14	39.6
February 29	39.2
Average	39.4
March 1	39.6
March 5	39.5

[a] Average of five highest maxima.

TABLE VIII.—*Highest daily maximal body temperatures. Acclimatization (monkey 4 kept in the sun and in contact with the heated roof)*—Cont'd.

1912.	°C.
March 12	39.4
March 16	39.8
March 30	39.5
Average	39.6
April [b] 10, 11	39.5
April 12, 16	39.5
April 18	39.9
April 22	39.5
April 26	40.1
Average	39.7

[b] During April the required hours for civil service employees were from 7.30 in the forenoon to 12.30 in the afternoon; hence the monkeys were not usually exposed much beyond noon in this month. Only on days that were expected to be very hot were the monkeys kept out in the afternoon. More will be said about this later.

It will be seen from Table VIII: (1) That never after December 1, the first day on the ground, was the temperature of the monkey found above 40.5°—that is, the combined action of the hot sun and hot roof on the hottest days did not produce as much response in the monkey as did the sun's rays alone on November 29, 1911 (a body temperature of 40.5° apparently produces no discomfort in the acclimatized monkey); (2) that never but once after December 15, the ninth day on the ground, did the temperature exceed 40°, which is a temperature but little, if any, above the maximal shade temperature of this monkey; (3) that the monkey's temperature never exceeded the maximal shade temperature in February and but once, if at all, in March, and but twice in April, the record hot month. This seems to make it clear that a great deal of the acclimatization took place in the first two days of exposure, November 29 and December 1, that by December 18, the eleventh day on the ground, a rather high degree of resistance had been acquired, and that by the end of the second month the resistance had probably reached its maximum. It also seems to show that the temperature of a thoroughly acclimatized monkey, exposed day after day to the sun in contact with a hot roof, rarely exceeds its maximal normal temperature.

The history of the monkey thus far indicates in a decisive manner, when the earlier history is compared with the later and when it is compared with the experiments of Aron: (1) That

there is a marked difference in the effects of the climate of Manila on different days; (2) that the maximal normal temperature of this monkey is probably not below 39.5°; (3) that this monkey had acquired increased power of resistance to the tropical climate at its worst; (4) that the acclimatization was probably most rapid in the first two days; (5) that the acclimatization probably did not approximate completion much before the end of two months' exposure; (6) that an acclimatized monkey may show no discomfort in the sun of Manila even when sitting on the hot roof during the hottest days of the year; (7) that the temperature of an acclimatized monkey sitting on a bare, hot asphalt roof day after day in the hottest portions of the year in Manila may not rise above its maximal normal temperature (39.9°) oftener than once or twice a month; and (8) that the sun's rays alone probably have no injurious effect upon the monkey.

ACCLIMATIZED MONKEYS COMPARED WITH UNACCLIMATIZED

Although the comparison of the behavior of monkey 4 in the sun with the behavior of the monkeys studied by Aron seems to prove the acquirement of greatly increased resistance by the former, the data were too few to be conclusive, and it was necessary to expose it and other monkeys that had been exposed to the sun alongside of monkeys that had not been so exposed. Table IX gives the results of exposure to the sun and in contact with roofs of acclimatized monkeys alongside of unacclimatized.

It is readily seen from the facts shown in Table IX: (1) that the unacclimatized monkeys placed in the sun in contact with the hot roof may die in from forty minutes to six hours, depending upon the individual resistance and upon the climatic factors obtaining; (2) that ten exposures to the sun produced greatly increased resistance to the sun, but were not always sufficient to produce thorough acclimatization; (3) that twenty-one exposures produced a high degree of immunity if not thorough acclimatization; (4) that the temperature of an acclimatized monkey rarely exceeds the maximum normal temperature, even when exposed to the combined action of the sun and the hot roof; (5) that sickness reduces the resistance to heat or to heat combined with other climatic factors (see monkey 14, April 26); (6) that exposure in contact with the hot ground or roof may prove rapidly fatal when exposure 1 meter above the roof produces no symptoms (see monkeys 16 and 19, March 8 and 12), that is, much the greater portion of the injury to monkeys exposed as Aron exposed them seems to be due to the heat from the roof rather than to the sun's rays.

TABLE IX.—*Effect of the sun and a hot roof on acclimatized and on unacclimatized monkeys exposed simultaneously under the same conditions.*

Date.	Acclimatized.				Unacclimatized.				Remarks.
	Monkey.	Exposures.	Duration of last exposure.	Result, body temperature.	Monkey.	Exposures.	Duration of last exposure.	Result.	
			H. m.	°C.			*H. m.*		
Dec. 29, 1911	4	21	7 20	39.9	7	9	[a]6 0	Death	Time not exact.
					8	1	1 28	do	
					9	1	1 1	do	
					10	1	1 56	do	
					11	1	1 42	do	
Mar. 8, 1912	4	75	7 15	39.1					
	12	43	7 15	40.7	16	1	4 55	([a])	1 meter above roof.
	13	43	7 15	39.95					
	14	41	7 15	39.2	16	1	0 40	Death	Contact with roof.
	15	41	7 15	39.3					(cont.)
Mar. 11, 1912	4	77	6 45	39.3					
	12	45	6 45	39.6					
	13	45	6 45	40.6	18	4	4 0	do	
	14	43	6 45	39.5					
	15	43	6 45	39.8					
Mar. 12, 1912	4	78	7 15	39.4					
	12	46	7 15	39.5	19	3	2 45	([b])	1 meter above roof.
	13	46	7 15	39.8					
	14	44	7 15	39.3	[c]19	------	0 47	Death	Contact with roof.
	15	44	7 15	39.5					
Mar. 28, 1912	4	92	7 10	39.0					
	12	60	7 10	39.2					
	13	60	7 10	39.2	23	1	0 40	do	Do.
	14	58	7 10	39.5					
	15	58	7 10	40.1					
Mar. 29, 1912	4	93	7 15	38.8					
	12	61	7 15	39.0	17	2	0 45	(?)	1 meter above roof.
	13	61	7 15	39.2					
	14	59	7 15	38.7					
	15	59	7 15	38.8	-------	------	1 13	Prostrated and unconscious. Death 1 hr. 22 minutes later.	Contact with roof.
April 26, 1912	4	110	7 15	40.1	20	[d]6	1 33	Death	Do.
	12	81	7 15	39.6					
	13	81	7 15	40.0	21	[d]6	1 17	do	
	14	[e]79	7 15	([f])	22	10	5 9	do	
	15	79	7 15	40.0					

[a] Body temperature, 39.6°.
[b] Body temperature, 41° and 39.8°.
[c] Continuation of experiment with monkey 19.
[d] Some of these exposures were 1 meter above the roof.
[e] Monkey 14 had been sick for the last few days.
[f] Died in 4 hours and 37 minutes.

Nature of this acclimatization.—In this research facts have been brought out that give some information as to the nature of the acclimatization produced.

First, acclimatized monkeys sweat much more than unacclimatized monkeys when placed on a hot surface in the sun. Aron [6] had stated that monkeys have no sweat glands and I, not having had occasion better to inform myself on this point, was greatly surprised soon after I had begun these experiments on acclimatization to notice, while watching a monkey in the sun, small beads of sweat glistening in the hair of its forehead. After this I made systematic examinations of the surfaces of the monkeys' bodies in order to get some idea of the amount of sweating. I regularly found beads of sweat on the heads of acclimatized monkeys placed on the roof in the sun, if the roof was very hot. Sweat was found on the backs of the monkeys also, being greater in quantity on the upper back and gradually diminishing in quantity toward the tail. At the root of the tail comparatively little sweat was found. The palms were frequently found wet with sweat, and they often left wet imprints when lifted from the surface of the asphalt roof. On other parts of the body I found very little sweat. On some parts I did not succeed in finding any, but since the skin seems everywhere provided with well-developed sweat glands, it seems probable that there was insensible perspiration over the areas that did not show visible sweat. I found, therefore, that the acclimatized monkey sweats a good deal while resting on a hot roof in the sun. On the contrary, I found that the unacclimatized monkey sweats very little, if at all, under like conditions. This difference in perspiring between acclimatized and unacclimatized monkeys was very striking, so striking that one might be justified in inferring that it was because of this difference in sweating that the body temperature of the acclimatized monkey, under conditions that cause a rapid and fatal rise in the body temperature of the unacclimatized, does not rise above the maximum normal.

Secondly, it was found that a small dose of atropin sulphate caused a marked rise in the body temperature of an acclimatized monkey exposed to a hot sun. Atropin has been found to diminish the action of sweat glands by depressing the secretomotor nerve endings in the glands and so preventing nerve impulses from stimulating the secretory cells. Each time that I injected a small dose of atropin sulphate under the skin of an acclimatized

[6] Aron, op. cit., p. 110.

monkey resting on the roof in a hot sun, the sweating decreased and the body temperature began to rise almost immediately and soon reached such a dangerous height that it was deemed necessary to remove the animal to the shade to prevent prostration and death from heat stroke. A like dose of atropin produced no change in the body temperature of monkeys in the shade. In fact, after such doses given to monkeys in the shade, I saw no marked symptoms of any sort. These experiments with atropin strongly support the inference already drawn that the difference in the amount of perspiring in acclimatized and unacclimatized monkeys is enough to account for the fact that unacclimatized monkeys die of heat stroke under conditions that do not raise the temperatures of acclimatized monkeys above the normal.

Thirdly, it was observe that when the relative humidity of the atmosphere was much higher the tendency to rise of the body temperature of an acclimatized monkey in the sun on the roof was markedly greater. Since an increase in relative humidity has a tendency to raise the body temperature by diminishing the rate of evaporation of water from the surfaces of the body, this supports the inference that it was the sweating of the acclimatized monkey which caused its temperature to remain normal under circumstances that killed the unacclimatized monkey with heat stroke.

Fourthly, attempts to acclimatize rabbits failed signally. The methods that had proved successful in acclimatization of monkeys were tried with rabbits. Rabbits were carefully exposed to the sun day after day, being removed to the shade as soon as they seemed approaching danger of heat prostration. The rabbits remained in good condition, but no increase in resistance to the action of the heat could be detected. On succeeding days the period of exposure could not be appreciably lengthened with safety to the animal. Rabbits that had been submitted to this process of acclimatization for many days in succession were exposed alongside of rabbits that had not been previously exposed. I was not able to detect any difference in the powers of resistance between the rabbits that had been submitted to the acclimatizing process and those that had not been previously exposed. The rabbits that had been submitted became prostrated as soon after insolation as the unexposed and died as soon. Rabbits are not known to perspire. Here there was no perspiring to be increased, and no acclimatization resulted. This fact, therefore, also gives indirectly some support to the infer-

ence that the acclimatization observed in the case of the monkey consisted in the development of increased capacity for sweating under the conditions of the experiment.

Mechanism of this acclimatization.—It remains to inquire how this increased sweating observed in acclimatized monkeys was brought about. It followed upon simple exposure to the sun, which means exposure to heat and light rays. The increase in sweating occurred upon the surfaces most exposed to the heat, namely, the palms of the hands, which came in contact with the hot roof, and the back, which was uniformly turned toward the sun. The primary effects of the heat upon the monkey were, first, the elevation of the temperature of that portion of the skin which was exposed to the heat rays, including, among other structures there found, the sensory nerve endings, the secretory cells, and the secretomotor nerve endings in the sweat glands; secondly, the elevation of the temperature of the entire body, most marked in the first few days of exposure. A secondary effect evidently was an increase in sensitiveness of one or more parts of the sweating mechanism, which increase was more or less persistant. Since a small dose of atropin stops the sweating in an acclimatized monkey, presumably by paralyzing the secretomotor nerve endings in the sweat glands, the increase in sensitiveness must lie in the nervous mechanism and not in the secretory cells of the sweat glands. The three portions of this nervous mechanism which seem most likely to be affected are the sensory heat nerve endings, the secretomotor nerve endings in the sweat glands, and the nerve cells concerned with temperature regulation. The endings mentioned would be most affected by the direct heating of the skin. The cells could be affected in two ways: First, by the arrival of a greater number and of stronger impulses, coming from the highly heated skin, and secondly, by being heated above their normal temperature by the increase in body temperature. Since, when stronger and more frequent impulses pass into a given tract of the brain, the brain becomes so changed that stimuli to this tract set up greater responses than before, it does not seem unreasonable to suppose that the nervous mechanism regulating body temperature, excited by the unusually strong heating of the skin, may become so changed that any nerve impulses coming to it will produce greater discharges than before. It may then be justifiable to assume that the acclimatization observed in these experiments consisted, at least in part, in a sensitization of nerve cells concerned with temperature regulation of which cells those con-

cerned with the stimulation of the sweat glands seem to be especially affected.

However, it must not be forgotten in this connection that there may have been other conditions operating to raise the resistance of these monkeys to heat. For example, the change in diet to which each monkey was subjected when it was placed under observation may have aided in increasing its resistance. Observations by the author in connection with experiments on man indicate that in man some diets lower the resistance to heat and others raise it. Whether or not the change in the monkey's diet was sufficient to affect its resistance remains to be ascertained. The fact that monkeys, partially acclimatized, kept in the shade on the same light diet, seemed to loose in powers of resistance indicates that the diet here plays a minor rôle.

The significance of this acclimatization of monkeys for the acclimatization of man.—Since a moderate ability to sweat and a light, suitable diet are sufficient to render the monkey practically immune to the worst combinations of weather conditions that occur in a tropical region such as that in which Manila lies, it would seem that man might readily be acclimatized to the same conditions. There are many facts that point in this direction. Man seems in every way better adapted than the monkey to resist the tropical climate. The monkey is ordinarily easily killed by heat stroke as has been shown by the experiments of Aron and by the experiments here reported. It may be, as Aron stated, that the monkey is less resistant to the worst combinations of tropical weather than the white man. The temperature-regulating mechanism in man is much more sensitive than that in the monkey, as shown by the smallness of the normal variation of body temperature in man as compared with that in the monkey. The sweating mechanism in man has many times the capacity of that in the monkey, and as the temperature of the surrounding atmosphere approaches the temperature of the body, this becomes the most important means of eliminating heat from the body. The internal heat production of man on a light diet is smaller in proportion to body surface than that of the monkeys subjected to this experiment. Man stands higher above the hot surface upon which he rests or moves; hence he would received less heat from the ground and be subjected to a more rapid movement of air over the body surface than would the monkey. Man's body has much less hair than the monkey's; hence the escape of heat from his body by radiation, conduction, or evaporation is less interfered

with. Man can so select and arrange his clothing that it will interfere little with the escape of heat from the body, while at the same time it will shield the body from the heat rays coming from the sun. Moreover man is acquainted with a larger variety of foods. This enables him to avoid the useless production of heat and toxins in the body. By taking at each meal only enough of each food to furnish the quantity of each element and each compound needed by the body at the time, he avoids for his body the necessity of burning an excess of any one food in order to get a sufficient quantity of some one of its contituents; he also avoids leaving a large or dangerous residue in the intestine, thus avoiding or diminishing the elaboration of poison in the intestine by bacteria.

Experimental acclimatization of the white man to the tropics.—The foregoing observations by me on the acclimatization of the monkey to the injurious combinations of climatic factors that may arise in the tropics led me to institute some experiments on the white man to see whether or not the inference in regard to man could be experimentally justified. The experiments were begun upon me—a blonde American—and have already continued for a period of six months. As far as the experiments have gone, they have justified in every particular the inference drawn. I now frequently walk at a rapid, forced pace, bareheaded in the Manila sun at midday for more than an hour at a time, without causing more serious symptoms than a moderately abundant perspiration. At no time were more serious symptoms observed than a mild erythema solare, resulting in moderate desquamation of epidermis and some increase in tan. At no time was there any sign of a deficiency in sweating, nor was there ever any indication that the necessary amount of sweating was at all exhausting.

It was noted, however, that changes in diet produced marked changes in the power of resistance. Since diet has such a marked influence and since the white man sweats much more readily than the monkey, it seems that the major factor in the acclimatization of the white man is not the development of his capacity for sweating, but rather the proper regulation of diet. There are other factors of more or less importance for the white man, for instance, the development of tan on the portions of the body exposed to the sun.

Duration of this immunity acquired by monkeys.—A few experiments seemed to indicate that acclimatized monkeys lose their resistance if they are kept in the shade for a few days.

I hope to make a more detailed report of these experiments in a subsequent paper.

SUMMARY

1. The normal temperature of the Philippine monkey probably varies between 37.5° and 39.5° C.

2. Unacclimatized Philippine monkeys (genus *Pithecus*) exposed to the sun in Manila live or die of heat stroke in the course of from several minutes to several hours, depending upon the conditions.

3. The conditions making for a rapid death are: A hot sun; proximity of a large, hot surface, such as the ground or a roof; high relative humidity of the atmosphere; and a low wind velocity.

4. The death under these conditions is due to an accumulation of heat in the body.

5. The lack of any one of the above conditions may prevent death.

6. The effect of the sun's rays alone on this monkey is comparatively slight.

7. These monkeys on a suitable diet become acclimatized to the conditions mentioned, if exposure to the conditions is gradual.

8. A small dose of atropin will cause the death of an acclimatized monkey by stopping the perspiration.

9. The acclimatization consists in an increase in the sensitiveness of the nervous mechanism which regulates the body temperature. The increase in sensitiveness produces an increase in the rate of perspiration under the conditions named above, producing in this way what may be termed an immunity.

10. This immunity is readily lost if the monkeys are kept in the shade.

11. Some forms of sickness diminish the powers of resistance to the above-mentioned conditions.

12. Some toxins produced in the intestine lower this resistance.

13. Rabbits showed no increase in resistance when treated in the manner used to acclimatize monkeys.

14. Healthy white men may be readily acclimatized to the conditions named, that is, to the tropical climate at its worst.

15. The amount of sweating necessary to keep the body temperature of a healthy white man from rising above normal is not excessive, even when the man is doing considerable physical work in the midday sun in such a tropical climate as that which obtains at Manila, provided the man has been sufficiently long

on a suitable diet and introduces himself gradually into the work in the sun.

16. In the acclimatization of the white man the most important factor is the proper regulation of the diet.

17. The effects from the tropical sun seemed to be exactly the same as the effects from the sun in the United States; that is, no effects were seen or felt in these experiments that were different from what would have been expected under like circumstances in the northern portion of the United States.

THE INFLUENCE OF BILE UPON THE DISTRIBUTION OF CHOLERA VIBRIOS IN THE DIGESTIVE SYSTEM OF EXPERIMENTAL CHOLERA CARRIERS [1]

By Otto Schöbl

(*From the Biological Laboratory, Bureau of Science, Manila*)

The distribution of cholera vibrios in the alimentary canal of experimental cholera carriers has been studied in the early experiments.[2] It was found that the excretion of cholera vibrios in the fæces of successfully infected animals is irregular and not as frequent as one would expect. Out of 26 samples of fæces taken from 26 guinea pigs, only 2 were found to contain cholera vibrios at the time of examination. In another experiment a systematic examination of fæces for the presence of cholera vibrios was made in 18 guinea pigs. Out of 71 examinations of fæces taken from these animals at various times, only 14 tests were positive.

During the studies on the influence of bile upon the state of cholera carriers in experimental animals,[3] it was discovered accidentally that animals fed on bile excreted cholera vibrios in their fæces more frequently than normal cholera carriers. This finding was considered important enough to be further studied.

A series of guinea pigs was infected by intravesicular injection. Some of the animals were fed on bile; the remainder were kept as normal controls. They were killed and examined for the presence of cholera vibrios at intervals. Cultures were made from the gall bladder, duodenum, ileum, cæcum, and fæces. Both direct Dieudonné's plate and peptone cultures were planted.[4]

[1] Received for publication October 5, 1916.

[2] *Journ. Inf. Dis.* (1916), **18**, 307–314; (1916), **19**, 145–152.

[3] *This Journal, Sec. B* (1916), **11**, 157.

[4] For details of technic, see *Journ. Inf. Dis.* (1916), **18**, 307–314.

TABLE I.—*Showing the influence of feeding bile upon the distribution of cholera vibrios in the alimentary canal of experimental cholera carriers as compared with that of normal cholera carriers.*

[+, cholera vibrios found; —, cholera vibrios not found; v. f., very few, less than 6 colonies; f., few, about 12 colonies; n., numerous, about 200 colonies; v. n., very numerous, more than 200 colonies.]

CARRIERS: RECEIVED OX BILE PER OS.

Guinea pig.	Examined, days after inoculation.	Direct plates.				Peptone cultures.				
		Bile.	Duodenum.	Ileum.	Cæcum.	Gall bladder.	Duodenum.	Ileum.	Cæcum.	Fæces.
1	8	v. n.	v. n.	v. n.	n.	+	+	+	+	+
2	8	n.	n.	v. n.	f.	+	+	+	+	+
3	9	n.	n.	v. n.	n.	+	+	+	+	+
4	9	v. n.	n.	v. n.	f.	+	+	+	+	+
5	10	v. n.	v. n.	v. n.	n.	+	+	+	+	+
6	12	v. n.	v. n.	v. n.	v. n.	+	+	+	+	+

CONTROL CARRIERS: RECEIVED NO OX BILE.

7	8	n.	v. f.	f.	v. f.	+	+	+	+	+
8	9	n.	v. f.	v. f.	—	+	+	+	+	+
9	9	n.	v. f.	v. f.	—	+	+	+	—	—
10	10	v. n.	v. n.	v. n.	f.	+	+	+	+	—
11	12	v. f.	—	v. f.	—	+	—	+	—	—

These experiments show that the distribution of cholera vibrios in the alimentary canal of experimental cholera carriers fed on ox bile was more extensive, the elimination of cholera vibrios in the fæces was more constant, and the cholera vibrios were present in the alimentary canal in larger numbers than in the case of the carriers that received no bile.

COMMON INTESTINAL PARASITES

By Faustino Garcia
(*From the Southern Islands Hospital, Cebu, P. I.*)

To obtain an idea of the prevalence of infection by intestinal parasites among Filipinos in the Visayan Islands of the Philippine Islands, the records of stool examinations from April, 1913, when the Southern Islands Hospital was established, to November, 1916, were examined. For these three and a half years the stools of 1,603 patients were submitted to routine examination. The majority of the patients were of the lower classes, so that my statistics represent more or less the common Filipino people residing in the Visayan Islands and especially in Cebu Island.

Table I gives the names of the intestinal parasites and the percentages of infections by the common intestinal parasites among the Filipinos whose stools were examined.

Table I.—*Percentages of infections by the common intestinal parasites among Filipinos examined.*

	Number.	Percent.
Patients examined	1,603	
Patients infected	1,066	66.5
Infections:		
Trichuris	669	41.11
Hookworms	502	31.31
Ascaris	446	27.82
Monads	284	17.70
Amœba	17	1.31
Strongyloides	7	0.43
Tænia	6	0.37
Oxyuris	3	0.17
Hymenolepis nana	1	0.06
Total infections	1,945	121.32

These results are not to be taken as the exact percentages of intestinal infections. It is to be admitted that each percentage of intestinal infection found represents the minimum obtained, for although two or more cover-glass preparations were made, still some might have escaped diagnosis.

TABLE II.—*Comparison of infections by the common intestinal parasites with other reports.*

Year.	Authority.	Country.	Examinations.	Infections.		Individuals infected.	
					P. ct.		*P. ct.*
1893	Dobson[a]	India	1,249	1,340	107.28		
1900	Fearnside[a]	do	878	921	104.90		
1901	Colvert[a]	do	100	143	143	92	92
1904	Anemia Commission[a]	Porto Rico	4,482	6,259	139.64	4,482	100
1908	Garrison[a]	Philippine Islands, Bilibid Prison.	4,106	7,636	186	3,447	84
1915	P. H. S.[b]	do	[c]13,297				67.7

[a] Garrison, *This Journal, Sec. B* (1908), **3**, 194.

[b] Report of the Philippine Health Service for the year 1915 (1916), 16.

[c] Fæcal specimens.

It will be seen from Table I that the number of infections is slightly greater than that found by Dobson or by Fearnside, but much less than that obtained by Garrison. The figures given by Colvert and by the Anemia Commission are not good for comparison, as the former examined only 100 persons, while the latter's work was for hookworm only and, consequently, not a true statistical statement of infections by intestinal parasites for the general population. The number of infected persons is about 70 per cent less than that obtained by Garrison, but almost equal to that reported by the Philippine Health Service in its annual report for 1915. The Filipinos examined by Garrison in Bilibid Prison in 1908 came from different parts of the Islands, and the Philippine Health Service report was for prisoners examined in 1915. Patients of the Southern Islands Hospital (1913–1916) came from the Visayan Islands—mostly from Cebu Island. How can we account for the great difference in results? The report of Bilibid Prison Hospital for 1915 shows 66.68 per cent, or about 1 per cent more than my findings here for patients admitted from 1913 to 1916. For this reason these data may be taken as the true statistical intestinal infection of the Filipinos for the period 1913 to 1916. That the number of infections is less than that obtained by Garrison in 1908 may be due either to the improvement in general sanitary conditions or to the education of the people with regard to personal hygiene.

GEOGRAPHICAL DISTRIBUTION

Attempts were made to find the infections by localities. In this case the last residences mentioned by the patients were taken as their true residences. The number of infected persons in each locality and the infections by *Trichuris*, hookworms, and *Ascaris* alone are compared.

TABLE III.—*Infections of patients by the common intestinal parasites in the Southern Islands Hospital.*

Locality.	Individuals.			Infected with—					
	Examined.	Infected.		*Trichuris.*		Hookworms.		*Ascaris.*	
			P. ct.		P. ct.		P. ct.		P. ct.
City of Cebu	728	477	65.52	326	44.77	187	22.68	223	30.63
Towns of Cebu Province	691	468	67.72	270	39.07	261	37.77	176	25.47
Cebu Province, including the city of Cebu	1,419	945	66.59	596	42.00	448	31.57	399	28.11
Outside of Cebu Island	184	121	65.76	73	39.67	54	29.54	47	25.54

Table II shows that the percentage of infected persons varies from 65 to 67 and that of *Trichuris* from 39 to 44. Hookworm and *Ascaris* infections vary greatly. In Cebu city, hookworm infection is 12 per cent less than in the towns of Cebu Province, while it is 5 per cent more than that of *Ascaris*. If we believe that the sanitary condition of the city of Cebu is better than that of other towns of Cebu Province, we can deduce that hookworm infection is greatly influenced and lessened by improvement of sanitation and *Ascaris* infection is altered only slightly, if at all. The individual infections of *Trichuris*, hookworm, and *Ascaris* in Cebu Province and outside of Cebu vary only from 2 to 3 per cent.

DISTRIBUTION BY SEX

Out of 1,603 patients examined, 354 were women. The following gives their corresponding infections.

TABLE IV.—*Infections of males and females by the common intestinal parasites.*

Sex.	Individuals.			Infected with—					
	Examined.	Infected.		*Trichuris.*		Hookworms.		*Ascaris.*	
			P. ct.		P. ct.		P. ct.		P. ct.
Males	1,249	819	65.58	496	39.71	423	33.86	328	26.26
Females	354	247	69.77	173	48.87	79	22.31	118	33.61

The females showed about 4 per cent more infections than the males. Infections with *Trichuris* and *Ascaris* were more numerous in the females; infections with hookworm were more numerous in the males. A greater percentage of hookworm infection in men than in women is to be expected for the reason that

infection of hookworm takes place mostly through the skin, especially of the legs and feet, and as a consequence the men, whose work is mostly out-of-doors, are naturally more liable to infection than women.

DISTRIBUTION BY AGES

To obtain an idea of the distribution of infection by ages, I divided the persons examined into decades from the first to the sixth. The seventh decade and over are included in the sixth decade. Table V shows the result.

TABLE V.—*Infestation by ages by the common intestinal parasites.*

Decade.	Individuals.			Infected with—					
	Examined.	Infected.		*Trichuris.*		Hookworms.		*Ascaris.*	
			P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
1st	98	63	64.28	43	44.08	14	14.28	42	42.85
2d	355	274	77.18	176	49.85	146	41.12	119	33.52
3d	615	393	63.79	242	39.12	197	30.35	145	33.54
4th	241	153	63.48	97	40.24	72	29.87	65	26.75
5th	164	99	60.36	55	33.53	42	25.61	42	25.61
6th and 7th	129	84	65.12	66	43.41	41	34.11	33	25.58

The greatest infection percentage is in the second decade, while the least is in the fifth. The variation is not great even in the case of *Trichuris*. In the case of hookworm and *Ascaris* differences are marked in the first and second decades. In the first decade hookworm infection is the lowest. In ascariasis the greatest percentage of infection in the first decade may be explained partly by the fact that children are fed by the mothers, who may be infected and whose personal hygiene is very limited. Unsanitary conditions of the house and surroundings may largely be contributing factors in the production of this greater percentage of ascariasis in the first decade. These facts might suggest that more instruction in taking care of children be given to the mothers.

SYMPTOMATOLOGY AND PATHOGENICITY

Trichuriasis.—The presence of *Trichuris trichiura* in the intestine has never been recognized in this hospital by any characteristic symptom that it may have produced, but only by finding the ova by microscopic examination of the stool. Although many consider that whipworm infection is of little importance, a review of the literature on this subject indicates that, if it is not patho-

genic in many cases, still it aids greatly in the aggravation of other diseases. That the whipworm is pathogenic in some cases has been illustrated in the report of Musgrave, Clegg, and Polk. [1]

Four detailed cases of trichocephaliasis were mentioned: two producing severe progressive secondary anæmias followed by death in one and no improvement in the other after one month of treatment; the third producing the symptoms of diarrhœa, muscular cramps, dizziness, œdema, and indigestion; and the fourth a variety of symptoms, which on autopsy showed no cause other than an embolism of the left coronary artery by an adult *Trichuris trichiura*. The above illustrations and the fact that *Trichuris trichiura* has been found in the appendix after operation at the Southern Islands Hospital, which confirms the statement in several textbooks [2] that it occasionally causes appendicitis and wounds which serve as entrance for microörganisms into the blood stream, indicates that more attention should be paid to whipworm infection than it receives at present.

Ankylostomiasis.—In the great majority of cases the presence of hookworm is demonstrated solely through the routine stool examination of every patient admitted to the hospital rather than because of any symptom of its presence. All the symptoms produced by hookworm infection as described in the textbook of Castellani and Chalmers are seldom observed in the Filipinos admitted to the Southern Islands Hospital. Anæmia was seldom present with hookworm infection among Filipino patients, and those who were anæmic were mostly so due to malarial cachexia, tuberculosis, or some other cause.

We have had several cases, however, which we diagnosed as true cases of ankylostomiasis. In these cases our diagnosis was based upon the history of eating nonedible materials, such as earth and coal, feeling of laziness of the patient, and the symptoms of secondary anæmia. On examination of the stools diagnosis in all these cases was confirmed.

Ascariasis.—The infection of *Ascaris* produces mostly no symptoms among Filipinos, and if it does, the symptoms are very variable and not characteristic. One symptom that may be suggested of *Ascaris* infection is a dull abdominal pain which may or may not be accompanied by fever. Few cases of this complaint were observed in this hospital that could not be attributed to causes other than that of infection by *Ascaris* and in which

[1] *This Journal, Sec. B* (1908), 3, 545.

[2] Castellani and Chalmers, Manual of Tropical Medicine, 2d ed. William Wood & Company, New York (1913), 1301.

treatment of *Ascaris* entirely relieved the pain. Fever simulating the beginning of typhoid has been observed in many cases with *Ascaris* infection, in which, after administration of santonin and calomel, the temperature returned to normal. Whether the fevers of three or six days, which disappeared after treatment of *Ascaris*, were due to *Ascaris* infection is hard to tell, but the fact is that after removing *Ascaris* in some of those cases the patients were cured. In children convulsions and fever were observed which, after administering appropriate treatment, ceased with the subsequent recovery of the patients. Secondary anæmias due to infection with *Ascaris* are also not uncommon.

In general, it may be said that Filipinos seem to be more or less immune to the effects of *Ascaris* and hookworm infections. Even in children I have observed 35 and 42 *Ascaris* worms expelled, in one of 5 years of age and the other of 3 years of age. Both of these children were well developed and well nourished without any symptoms of intestinal parasites. They were admitted to the hospital as cholera carriers, and infections of *Ascaris* and hookworm were discovered only upon routine stool examinations.

TREATMENT

The drugs that are advocated by many authors for *Ascaris* and hookworm infections are the ones that have been used in the routine treatment of ascariasis and ankylostomiasis. The following order has been given for hookworm treatment:

Nothing to eat; stop all medicine and give magnesium sulphate saturated solution, 40 cubic centimeters at once.

After three hours give thymol, grains XL, one dose. Followed in two hours by magnesium sulphate saturated solution, 40 cubic centimeters.

Liquids may be given after free purgation.

Following day give usual treatment diet.

The following order was instituted in the treatment of ascariasis:

Give at one dose the following:

Santonin	grains V
Calomel	grains II
Sodium bicarbonate	grains V

Follow in five hours with magnesium sulphate saturated solution, 40 cubic centimeters.

The above treatments serve their purpose, but they are not ideal, as shown by the fact that in most cases the treatments were repeated many times before a complete elimination of the worms

resulted, as shown by the negative microscopic findings of the stool.

To observe the effects of broken doses, the same drugs with the same amounts were given as follows:

Nothing to eat; stop all other medicine and give magnesium sulphate saturated solution, 40 cubic centimeters.

After two hours give thymol, grains XL, in four doses at half-hour intervals.

After the last dose, repeat magnesium sulphate saturated solution.

For *Ascaris* treatment the following was substituted:

Give the following formula in four broken doses, half hour apart:

Santonin	grains V
Calomel	grains II
Sodium bicarbonate	grains V

After three hours of last dose, give magnesium sulphate saturated solution, 40 cubic centimeters.

These broken doses were given for two months, and the effects were compared with those in which the drugs were given in one dose for two months also.

TABLE VI.—*Comparison of effects of one dose and of broken doses.*

ASCARIS.

Results of one dose.		Results of broken doses.	
Persons treated.	Treatments given to produce negative finding for ova.	Persons treated.	Treatments given to produce negative finding for ova.
4	2	6	2
2	3	2	3
1	6	1	4
1	7	1	7

HOOKWORMS.

3	1	11	1
3	2	1	2
2	4		

From the tabulation it will be seen that the results of the treatment of *Ascaris* did not vary greatly, whether the formula was given in one dose or in four broken doses. As to hookworm treatment, it seems from the observation of the 8 and 12 cases that the broken doses are more effective in eliminating the hookworms than when giving the thymol in one dose.

SUMMARY AND CONCLUSIONS

The total intestinal infections among the Filipinos in the Visayan Islands examined in the Southern Islands Hospital is 121 per cent.

The percentage of infected individuals is 66.5.

The *Trichuris* infection alone is 41 per cent.

The hookworm infection alone is 31 per cent.

The *Ascaris* infection alone is 27 per cent.

Ascaris infection is greater in the city of Cebu than in the towns of Cebu Province, while the reverse is true with hookworm infection.

The second decade has the greatest percentage of infection.

The symptoms of intestinal infections among Filipinos are indefinite, and it seems apparent that the Filipinos are not very susceptible to intoxication of hookworm as compared with the white race.

The treatment of *Ascaris* infection with santonin and calomel, though it serves the purpose of eliminating the worms after several treatments, is not ideal.

The treatment of hookworm infection is more effective when the thymol is given in broken doses than when given in a single dose.

A CASE OF ADVANCED PREGNANCY IN THE BROAD LIGAMENT [1]

By Potenciano Guazon

(From the Department of Surgery, College of Medicine and Surgery, and the Philippine General Hospital, Manila)

F. G., 38 years old, Filipina, housewife, born and residing in Manila, was admitted to the surgical service of the Philippine General Hospital complaining of enlargement of the abdomen. Her father and mother died of diseases unknown to the patient. Her husband is living and well. The patient denies having had venereal diseases. There was no history of tuberculosis or tumor.

She had smallpox in childhood. Has occasional headache and fevers of short duration. Other diseases are denied.

Menses began at the age of 17 years. They have been regular, of about three days' duration, without any accompanying symptom till the sixth pregnancy, when she aborted, and afterward her menstruation became scanty in amount, lasting only for one or two days and always accompanied by pain transmitted to the upper region of the abdomen. She denies having any vaginal discharge.

The patient has had eleven normal deliveries. No history of puerperal infection was elicited. As stated above, she aborted her sixth pregnancy at the third month. At this abortion she had continuous bleeding for two weeks, which was at first profuse and gradually decreased in amount until it disappeared spontaneously. Her last delivery took place five years ago, being at full term and perfectly normal. Every time she became pregnant she had severe morning sickness lasting for two or three months. During this time she usually became nauseated and could not eat anything except sour food.

Five months ago she felt paroxysmal pain in the lower abdomen, usually at night. Later she noticed a tumor mass growing in the same region, which gradually increased in size and was slightly painful at times. During these months her menses stopped entirely, but she did not notice any other symptoms of

[1] Read before the Manila Medical Society, April 3, 1916.

pregnancy, except that she was becoming fleshy and her breasts were slightly enlarged, though painless and not containing milk. Her appetite has been usually good. The bowel movements are regular.

PHYSICAL EXAMINATION

The patient is well developed and nourished and is able to be about without discomfort. The sensorium is clear. Examination of the body reveals nothing important except the following:

Chest. The breasts appear slightly enlarged, but no secretion could be obtained from them, even on strong pressure. No other signs of importance.

Abdomen. There is a bulging occupying almost the whole lower part, reaching to midway between the umbilicus and symphysis pubis. It appears globular and free from the anterior abdominal wall. It is movable and can be pushed in all directions. There is slight fluctuation; the sensation is much like that of a fluid under high tension. The mass measures about 20 by 20 centimeters and is not tender on pressure. The greatest circumference of the abdomen passing over this tumor is 93 centimeters.

Vaginal examination. The perineum is lacerated in the second degree. The vaginal mucosa is of normal color. The cervix is soft, slightly œdematous, and follows with the mass described above, when moved. The body of the uterus cannot be felt due to the pressure of the tumor.

As the history given by the patient is somewhat misleading, the diagnosis of ovarian cyst or some kind of tumor was suspected by the resident surgeon in charge of the case before operation.

OPERATION

The patient was operated upon by me. A low median incision was made. On opening the peritoneum, a tumor mass was encountered filling the lower abdomen, rather toward the left iliac region. Its surface is covered with numerous dilated vessels. It was of uneven consistence, soft in some parts and firm in other places. A cannula was introduced, but pure dark blood only was obtained. On manipulation, a piece of cauliflowerlike tissue came out and a suspicion of papilloma was then entertained; but while shelling off the tumor from the folds of the left broad ligament, a hairy surface appeared. Then the mass was opened, and a fœtus of about five months was found.

The fœtus was between the folds of the broad ligament displacing the uterus, which was enlarged and soft, toward the right side. The placenta was shelled out entirely by the fingers.

The bleeding, which was considerable, was stopped partly by pressure and partly by means of ligatures. The raw surface of the uterus was covered up, and the abdomen was closed without drain.

The patient had no postoperative complications and was discharged in good condition two weeks after the operation.

PATHOLOGIC EXAMINATION OF THE PLACENTA AND OTHER TISSUES REMOVED WITH IT

A careful examination of the specimen showed that a portion of the placenta was firmly attached to the fallopian tube and ovary. The case can be interpreted, therefore, as one of tuboovarian pregnancy at the beginning, which became intraligamentous later, following a rupture into the folds of the broad ligament.

DISCUSSION

The case related here and other similar cases of ectopic pregnancy are generally admitted as relatively uncommon gynecological affections. Because of their infrequence and lack of definite pathognomonic symptoms, they are not easily diagnosed and are very often mistaken for other pathologic conditions.

If it is true that, in the reported case, menstruation had been absent for about five months, the same condition may also occur, however, in an ovarian cyst, although such is not the rule. Besides, the patient is almost at the period of menopause, being 38 years of age.

The enlargement of the breasts alone, without the presence of other signs accompanying an ordinary pregnancy, may take place also in certain genital tumors.

The œdematous appearance of the cervix might have been caused, just as well, by pressure from the tumor mass, which displaced the uterus to one side. This same pressure and displacement producing passive congestion may also cause enlargement of the uterus.

Auscultation was not employed in the case, but as the fœtus was delivered apparently dead, its result would have been probably negative.

However, in diagnosing a case of ectopic gestation, we must take into account that the symptoms and signs usually found in an ordinary pregnancy are frequently obscure or wanting in the former affection, and for this reason it is advisable not to neglect any findings that may be obtained from history or from physical or laboratory examination which may point to the suspicion of such a condition.

In the reported case, for instance, such findings as cessation of menstruation corresponding to the period of growth of the abdominal swelling, fullness of the breasts, soft, œdematous cervix, and a period of sterility for five years may be taken as sufficient data to arouse suspicion of the disease. But as this affection is not as common as tumors of the ovary and uterus, such as ovarian cysts, fibroids, etc., the exact condition is not as a rule diagnosed.

I recall that in the surgical service of Doctor Davis, in the Philippine General Hospital about three years ago, there was a case of advanced extra-uterine pregnancy which was diagnosed as tumor before operation. Nearly two years ago Roxas(6) reported to the Colegio Medico-Farmaceutico two cases of abdominal pregnancy (secondary) at full term, from the obstetrical service of Doctor Calderon in the same hospital. One of them was not diagnosed as such, but, apparently, as a case of intra-uterine pregnancy. It is to be observed that both patients came to the hospital calling for delivery.

The proportion of extra-uterine to intra-uterine pregnancies cannot be exactly determined, as statistics vary according to individual reports and those coming from the different clinics. According to Hirst,(2) it is given by some writers as being 1 to 500 approximately. From 1908 to 1915, in our surgical service, we had about 40 cases of ectopic pregnancy which, compared with the 5,000 cases of delivery registered in the obstetrical department of the College of Medicine and Surgery, during the same period, will show a proportion higher than that mentioned above.

The majority of the cases of ectopic gestation do not reach an advanced stage,(1) as they become ruptured or aborted during the early months of their development, resulting in the destruction of the fertilized ovum. Taking, for example, tubal pregnancies, which are the commonest types of ectopic gestation, according to Winkle(3) about 6 per cent of them only may proceed to full term. Hirst(4) reported two cases of tubal pregnancy that ruptured in two weeks. In our series of 65 cases, 2 progressed to full term (Calderon and Roxas), and there are 2 others that reached only the age of four to five months (Davis and Guazon).

In regard to treatment of advanced ectopic pregnancy, some gynecologists advise not to interfere until full term or until the stage of viability has been attained; while others do not distinguish any condition and operate as soon as the diagnosis is suspected or established. I see no objection to an early inter-

vention, if we consider that this kind of gestation is an abnormality and consequently may endanger the life of the patient at any time. We know also from statistics that a great number of fœtuses or babies delivered from extra-uterine pregnancies are found either dead or deformed or live only for a short time.(5) In our case, like Dr. Davis's, the fœtus appeared deformed and was delivered without signs of life. One of Doctors Calderon and Roxas's cases was also deformed and survived only for about ten minutes.

As to the management of the placenta, there is again a difference of opinion, as there are operators that favor its removal in one or more sittings. If the placenta can be removed without causing a fatal hemorrhage or other serious complications, it should be always done, in order to shorten the convalescence of the patient. The technic of operation will depend much on the anatomical findings on exploration, so that the procedure will vary according to individual cases. In our particular case the sac and placenta were taken out completely, and the abdominal incision was closed without drain.

REFERENCES

(1) Guazon, P. *Rev. Filipina Med. y Farm.* (1913), 4, 187.
(2) (3) (4) Hirst's Text-Book of Diseases of Women. W. B. Saunders Co., Philadelphia (1905).
(5) Horsley, J. *Surg., Gyn. & Obst.* (1913), 1, 58.
(6) Roxas, B. *Rev. Filipina Med. y Farm.* (1915), 6, 687.

ANNOUNCEMENT

Following an arrangement entered into between the Manila Medical Society and the Philippine Journal of Science, beginning with the year 1917, the latter will publish the minutes and abstracts of the proceedings of the meetings of the Manila Medical Society as evenly distributed as possible throughout the six issues of Section B, Tropical Medicine, of the Philippine Journal of Science. The Manila Medical Society submits its own copy. The arrangement is undertaken for the calendar year 1917 and succeeding calendar years until terminated in writing at the close of any year by either party. All original papers or articles read before the Manila Medical Society and bearing on the science of tropical medicine as shall be deemed by the Manila Medical Society to be worthy of publication in their entirety will be accepted for publication in Section B, Tropical Medicine, of the Philippine Journal of Science, as heretofore, under the same conditions as apply to articles submitted for publication from other sources. When an article is to be published in full in the Philippine Journal of Science, it will be referred to only very briefly or by title in the minutes and abstracts of the proceedings of the meetings of the Manila Medical Society. The pages of the minutes and abstracts of the proceedings are to be separately numbered and at the end of each year indexed in accordance with copy furnished by the society.

ALVIN J. COX,
General Editor,
Philippine Journal of Science.

Issued with THE PHILIPPINE JOURNAL OF SCIENCE, SECTION B,
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PROCEEDINGS OF THE MANILA MEDICAL SOCIETY

ANNUAL MEETING, JANUARY 6, 1917

On the evening of January 6, 1917, the Manila Medical Society embarked for the annual business meeting on the U. S. Army transport *Merritt* for Corregidor as the guests of the officers of the Medical Corps stationed on the island.

The following report of the proceedings of the meeting of the Manila Medical Society is taken from the minutes of the secretary, Lieut. H. G. Maul:

The regular monthly and annual meeting of the Manila Medical Society was held at Corregidor in the hospital building on the evening of January 6, 1917.

The meeting was called to order by the president, Dr. B. C. Crowell, who immediately appointed Dr. John A. Johnston, Dr. R. B. Gibson, and Capt. J. H. Trinder as a committee on nominations for the officers of the society for the ensuing year.

The minutes of the previous meeting were read by Dr. C. H. Manlove and were approved as read. Doctor Crowell then stated that arrangements were about to be completed in which the annual dues for membership would also entitle the members to Section B of the Philippine Journal of Science.

The nomination committee then returned and recommended for president, Lieut. Col. F. A. Winter, M. C., U. S. Army; for vice president, Dr. E. S. Ruth; for secretary and treasurer, Lieut. H. G. Maul, M. C., U. S. Army. There were no objections to the nominations, and it was moved and seconded to accept the recommendations of the committee. Prof. F. G. Haughwout then made a motion that the secretary be instructed to cast a unanimous ballot in favor of these nominations. It was seconded by Doctor Johnston. The new officers were then formally installed.

There then followed an informal scientific and social session and smoker. Dr. Paul Clements spoke of the work and the problems of the Philippine Health Service. Dr. N. M. Saleeby discussed the duties of the civilian practitioners and their relation to the military and naval medical corps. Major Rutherford gave an account of the case of anterior poliomyelitis lately discovered at Corregidor and enumerated in detail the quarantine measures that had been taken.[1] Other speakers in lighter vein

[1] See minutes of the subsequent meeting, February 13, 1917.

were Lieut. Col. J. T. Clarke; Col. C. L. Philipps, C. A. C.; Maj. W. M. Fassett, 13th Infantry; Lieut. Col. A. W. Kimble, Q. M. C.; Capt. Alex. Hall; and Capt. Franc Lecocq, C. A. C.

Professor Haughwout then moved that a vote of thanks be extended to the Quartermaster Corps, Colonel Crosby, Major Manly, Captain Trinder, the medical officers of Corregidor, and the program committee. The motion was seconded by Doctor Crowell. It was unanimously carried.

Doctor Gibson then moved that a vote of thanks be extended to the outgoing officers of the society. This motion was seconded by Doctor Johnston and was unanimously carried.

En route a business meeting of the Philippine Islands Medical Association was held, a report of which is as follows:

MINUTES OF THE PHILIPPINE ISLANDS MEDICAL ASSOCIATION

MEETING HELD JANUARY 6, 1917

In the absence of the president and both vice presidents, the meeting was called to order by the secretary-treasurer. On motion, seconded and carried, Councillor Saleeby was directed to take the chair.

The secretary-treasurer announced that the business of the meeting was the election of officers. On motion, duly seconded and carried, the association proceeded to the election of officers.

The nominating committee appointed by the chair presented the following names:

For President:	Dr. B. C. Crowell.
For First Vice President:	Dr. A. G. Sison.
For Second Vice President:	Dr. Thos. F. Keating.
For Councillor for five years:	Dr. S. V. del Rosario.
For Councillor for four years:	Dr. José Albert.
For Councillor for two years:	Maj. Clarence J. Manly, U. S. Army.
For Councillor for one year:	Capt. Charles C. Hillman, U. S. Army.

There being no further nominations, the secretary-treasurer was instructed to cast a unanimous ballot for the association for each of the above officers.

Doctor Crowell took the chair.

On motion to adjourn, duly seconded and carried, the meeting was adjourned at 8.20.

R. B. GIBSON,
Editor of the Proceedings,
Manila Medical Society.